



SOME ASPECTS OF EPILEPSY



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Clinical and diagnostic aspects of the epileptic syndrome of alcoholic origin

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Abstract

Alcohol epileptic syndrome is one of the most frequent neurologic manifestations of alcohol dependence occurring during withdrawal. The goal of the study was to investigate psychophysiological peculiarities of patients with alcohol epileptic syndrome in order to develop special diagnostic algorithms for its detection and selection of correct treatment and management approaches. The main methods of the study were screening, a clinical method, electroencephalography, computerized tomography, a laboratory method.

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Keywords: Alcohol epileptic syndrome; Alcohol withdrawal syndrome; Neurophysiological peculiarities; Clinical and psychophysiological diagnostic criteria; Alcohol-associated neurological disorders.

Abbreviations: ES: Epileptic Syndrome; AWS: Alcohol Withdrawal Syndrome; AES: Alcohol Epileptic Syndrome; AST: Alanine Aminotransferase; AST: Aspartate Aminotransferase; GGTP: Gamma-Glutamyl Transpeptidases; Aph: Alkaline Phosphatases; Cph: Creatine Phosphokinase; EEG: Electroencephalography; CT: Computerized Tomography; CAI: Chronical Alcoholic Intoxication.

Introduction

The tendency for increased use of strong drinks results in increased number of alcohol-associated somatic and neurological disorders including increased frequency of Epileptic Syndrome (ES) occurrence [1].

Epileptic attacks in Alcohol Withdrawal Syndrome (AWS) appear in connection with cancellation of alcohol use in the first 12-72 hours of abstinence [2]. In 90% of cases, epileptic syndrome of alcoholic origin don't require any special medical treatment with antiepileptic preparations or surgical interven-

tion in comparison with idiopathic (primary) or symptomatic (secondary) epilepsy [3].

The AWS psychophysiological mechanism is directly associated with dramatic alcohol withdrawal. It is known that on the neural level, ethanol starts up the following system brain processes: It reduces influence of GABA on chlorine canals, slows down action of glutamate on NMDA-receptors, that inhibits the excitation system and causes abnormal excitation processes, as well as increases brain concentration of adenosine, an inhibi-



tory neurotransmitter. Abnormal regulation of GABA-mediated inhibition and activation of NMDA-receptors account for excitation effect in dramatic alcohol withdrawal [4-6].

Differential diagnosis of Alcohol Epileptic Syndrome (AES) with idiopathic and symptomatic is very urgent.

The goal of our study was to detect specific neurophysiological peculiarities of patients with AES to increase effectiveness of ES differential diagnosis.

Tasks of our study were to detect electroencephalographic patterns and specific dynamic changes in electroencephalographic in patients with epileptic syndrome, to develop specific criteria the epileptic syndrome of alcoholic origin and to develop diagnostic algorithms of evaluation of results of clinical and psychophysiological examination.

There have been examined 251 persons (18-76 y.o.), their average age was 42.3 ± 0.89 . At the time of the study, they stayed at the Neurological Department of the Severodvinsk City Hospital, the Arkhangelsk region, Russia. In the course of the study, three groups of patients have been distinguished. I group included 89 patients with AES-with primary generalized attacks of wakefulness and AWS symptoms without the brain limited organic injuries in anamnesis, average age 41.66 ± 1.15 ; Of them, 16 persons- under 30, 67 persons-at the age 31-55, 6 persons - over 55; in II group- with non-alcoholic ES (78 persons), of them 26 persons - under 30, 29 persons-at the age 31-55, 23 persons - over 55. II-a subgroup included 22 persons, the average age 54.23 ± 3.04 ; of them, 2 persons-under 30, 7 persons-at the age 31-55, 13 persons-over 55, with-symptomatic- with primary generalized and secondary generalized attacks of sleep and wakefulness and the brain organic limited injuries (cerebrocranial injuries, insults, tumors in anamnesis) with AWS symptoms. II-b subgroup included 56 persons, the average age 36.98 ± 1.86 ; of them, 24 persons - under 30, 22 persons-at the age 31-55, 10 persons-over 55, with idiopathic-with primary and secondary generalized attacks of sleep and wakefulness without AWS symptoms. III gr. (comparisons) included 84 neurological patients, the average age 43.40 ± 1.66 ; of them, 25 persons-under 30, 39 persons-at the age 31-55, 20 persons-over 55, without ES and AWS.

The age difference among the patients with ES was statistically significant in I and II-a groups ($p < 0.001$), in I and II-b groups ($p < 0.05$), what confirmed the fact that ES of various genesis occurred frequently in different age groups. So, attacks manifested in childhood and at a young age, were more often caused by hereditary factors, perinatal pathologies, abnormal development, at a middle and late age- by tumors, vascular diseases, brain injuries, alcoholism. AES occurred more frequently in persons at a middle age. The age difference among the patients was insignificant in the I and III groups.

The following methods were applied during the study of frequency of occurrence of different neurophysiological characteristics: Screening -detection of sings of alcohol dependence and a high risk of its emergence, neurological examination of the patients, electroencephalography computer tomography, laboratory method for study of blood serum biochemical indices-Alanine Aminotransferases (ALT), Aspartate Aminotransferases (AST), Gamma-Glutamyl Transpeptidases (GGTP), Alkaline Phosphatases (APh), Creatine Phosphokinase (CPh) and statistical method [7-9].

Hereditary epileptic taint in anamnesis, attacks during sleep (more often at 4 and 8 a.m.), epiphenomena: Disorders of falling asleep, «deja vu», «meja vu», dreadful stereotypic colorful dreams, speaking in dreams, walking in dreams, febrile convulsions in childhood, a changed personality of epitype (circumstantiality, emotional viscosity, unbalance) were most peculiar to persons with idiopathic epilepsy, and a previous craniocerebral injury allowed to suspect symptomatic epilepsy. For intoxication ES, it was typical that patients abused alcohol, were registered at a narcologist's and had only daytime primary generalized attacks (attacks during sleep exclude intoxication genesis). During clinical examination of patients with ES with suspected intoxication, focal and meningeal symptoms were not detected, and polyneuropathy of lower extremities and abstinent syndrome occurred in 90% of cases. Alcoholic ES laboratory markers were increased activity of gamma glutamylpeptidase, aspartate and alanintransferase, if the level of bilirubin was allowable and liquor was not changed. GGTP was most active, its activity was tens times higher and in 3-5% of cases - a hundred times higher. The level of ALT activity rated second, it exceeded the maximum allowable level 10-30 times, AST activity in 7-10% of cases was only 2-3 times higher. Almost in 90% of cases on the 5th-6th day of hospitalization, GGTP and ALT indexes were 2-3 times lower with further reduction in a week period of time to normal or slightly higher than normal indexes.

EEG of all the patients in I gr. had the following accents: Aplanate or low-amplitude alpha-rhythm with absent zonal differences and modulations as well as regular dominating activity; diffusive changes in the form of slow waves from the delta range above both brain hemispheres, high frequency beta-rhythm primarily in frontal abductions; record plural artifacts; overlapping of "muscular tremor" and "floating electrodes" as AAS manifestation; epileptic and focal slow wave activity was not present on any EEG, but reduced activation after hyperventilation, photo- and phonostimulation has been detected.

82.0% of the patients from the I group had diffuse hypotrophic hydrocephaly on brain CT.

Mini-dictionary of terms

Alcohol epileptic syndrome (AES)

Epileptic attacks in appear in connection with cancellation of alcohol use in the first 12-72 hours of abstinence. Differential diagnosis of Alcohol Epileptic Syndrome (AES) with idiopathic and symptomatic is very urgent.

ECG

Recording was made on a 19-ported computer electroencephalograph "Neuron-Spectrum" with the use of the international lead system «10-20» with application of 19 active electrodes on the 1st-2nd day of a patient's admission to the Hospital, re-examination of some patients-on the 3rd-7th day.

Laboratory method

For study of blood serum biochemical indices-Alanine Aminotransferases (ALT), Aspartate Aminotransferases (AST), Gamma-Glutamyl Transpeptidases (GGTP), Alkaline Phosphatases (APh), Creatine Phosphokinase (CPh)-a biochemical auto analyzer «Vitalab Flexor E» was used. The study was conducted on the 2nd day of hospital admission. If enzymes' indexes were high, the study was repeated in 3-5 days.

CT test

On the 1st-3rd day of hospital admission, a computer tomograph with technology of helical scanning «High Speed Dxi General Electric» (US) was used. In the course of implementation of a layer-specific axial section test according to the international testing standards, brain sub- and supratentorial structures were analyzed. The width of scans (Tomograph step) was 1 cm, on brain base, it was 0.5 cm. If a mass brain lesion or any other local pathology was suspected, a contrast agent was administered for verification of obtained results.

Summary points

On the basis of the study conducted, it has been concluded that the patients suffering from AES had the following significant anamnestic and neurophysiological characteristics:

- Alcohol anamnesis, primary generalization of paroxysms, attacks of wakefulness,
- Absence of epiphenomena, personality disorders of epileptoid type, hereditary tainted epilepsy, craniocerebral injuries and insults in anamnesis; focal and general brain symptoms during clinical examinations,
- Absence of epileptic and focal activity on EEG,
- Presence of low electrobiological activity, diffuse changes of the brain, superposition of muscular tremor on EEG,
- Presence of mixed hypotrophic hydrocephaly on the brain CT,
- Blood serum transferases' increased activity and further rapid recovery within 2-3 weeks,
- Alcoholic polyneuropathy of lower extremities-a frequent concomitant pathology.

In cases of detection of epileptic activity on EEG, epileptic attacks can not be caused by alcohol. In those cases, alcohol was only an initiating agent.

Epileptic phenomena occur more often in patients with idiopathic epilepsy, who suffer neither from ES nor from CAI.

EEG changes typical of patients with AES practically do not

occur in patients with idiopathic or symptomatic epilepsy, as well as in persons who do not suffer from CAI or epileptic attacks. A common feature for the patients with nonalcoholic ES was typical changes on EEG in the form of epileptic or slow-wave activity.

Beside epileptic activity in the groups of the patients with nonalcoholic ES and without alcoholic ES and CAI, there were often observed changes on EEG like conditional-epileptic activity and dysfunction of medial-stem structures, that is not typical for the patients with AES [10].

Mixed hypotrophic hydrocephaly is a general sign indicating brain diffuse lesion; however in cases of CAI absence, it occurs in health only in elderly patients. Brain lesions caused by CAI result in CT-pattern of hypotrophic hydrocephaly in patients at the middle and even young age.

A correct classification of attacks and accurate detection of ES types will provide rational and individual basis for therapy, allow to improve prognosis and quality of life of patients suffering from different epileptic attacks.

Diagnostics of AES should include a total of anamnestic, clinical, laboratory and instrumental data. So, sings of «alcohol anamnesis», screening tests positive to alcohol dependence, absence of hereditary tainted epilepsy in anamnesis, epiphenomena and epileptic personality features do not mean that attacks can be only of alcohol origin. Focal or total brain and meningeal symptoms within the clinical pattern are typical for symptomatic epilepsy, and alcohol is often an initiating agent, but not a causative factor. Absence of those symptoms presupposes a possibility of intoxication or idiopathic attacks. Polyneuropathy of lower extremities as another significant sign of CAI is typical of patients with alcoholic epileptic attacks, but its occurrence together with a high level of blood serum transferases can not give a decisive answer about the reason of the epileptic attack. Immunological investigations will help to exclude another genesis of polyneuropathy of lower extremities (hereditary, autoimmunological, paraneoplastic, toxic). High activity of ALT, AST, GGTP is a biomarker of CAI [10]. Detection of epileptic activity means that the attack cannot be of alcoholic genesis. CT brain testing of patients who have had primary epileptic attacks is also necessary as a final link in the diagnostic process.

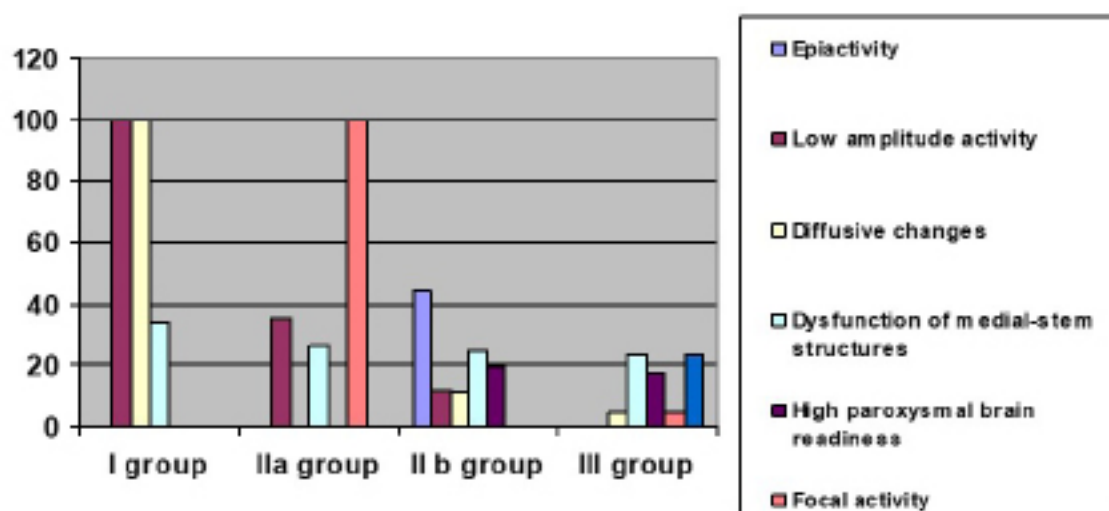


Figure 1: The frequency of neurophysiological indexes detected by use of electroencephalographic method in the studied group and the comparison groups (%).

EEG of the patients with AES besides the change of the amplitude-frequency indices towards slowing, applanation and irregularity of the main rhythm, diffusive onset of the excessive number of slow waves, were characterized by rarely occurring paroxysmal activity, absence of epileptic activity, decreased activation reaction to hyperventilation, photo- and phonostimulation, overlapping of "muscular tremor" and artifacts, what did not occur practically in the patients with idiopathic and symptomatic epilepsies, this can be used as an additional criterion of AES differential diagnosis. On EEG in 77.1% of the patients from the II-a group low-wave activity in brain injured hemispheres was determined, in 9.1% of cases- low electrobiological activity of the brain, in 3.6 %-dysfunction of medial-stem brain structures. On EEG of 44.6% of the patients, epileptic activity was detected in the form of commissures, discharges «peak- sharp wave», «sharp wave-slow wave»; in 25.0% - dysfunction of medial-stem structures; in 19.4% of the patients from the II-b group-high paroxysmal brain readiness in the form of hyper-synchronous sharp alpha-rhythm, discharges of bilateral synchronous sharp alpha-rhythm polyphase alpha-kin waves with an amplitude twice as high as the background, in 10.7% - low-amplitude type and slight diffuse changes were recorded. EEG of the patients from the III group in 23.8% of cases, there was revealed a dysfunction of medial-stem structures in the form of single discharges of bilateral synchronous polyphase alpha-kin waves with the amplitude up-to-background, flatness of zonal differences; in 17.9% heightened paroxysmal brain readiness (frontier EEG type) was detected; in 4.8% of cases focal slow wave activity was registered; in 23.8% there was no pathology detected.

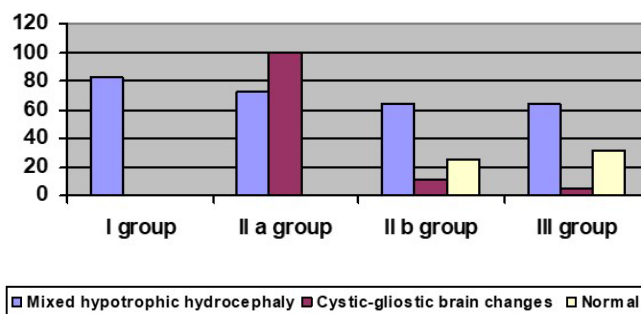


Figure 2: The frequency of psychophysiological indexes detected by use of computerized tomography method in the studied group and the comparison groups (%)

82.0% of the patients from the I group had diffuse hypotrophic hydrocephaly on brain CT. On CT of the brain of the patients from the II-a group in 100% of cases, cystic-gliostic brain changes were detected.

On CT of the brain, 25% of the patients from the II-b group had indexes within the norm, 10.7% of the patients had cystic-gliostic brain changes of posttraumatic nature, 64.3% of the patients had mixed hypotrophic hydrocephaly.

In the III group on CT of the brain in 31.0% of cases, there were no changes registered, in 64.3% - there were signs of mixed hypotrophic hydrocephaly, in 4.7% - cystic-gliostic brain changes were registered.

Table 1: The frequency of anamnestic and neurophysiological indexes detected by screening, use of clinical and laboratory methods in the groups of the studied patients (%).

Indexes	I group	II group		III group
		Ila	Ilb	
Epiphenomena	0	50	73.5	23.8
Hereditary tainted epilepsy	0	81.8	19.4	7.1
Cl, brain tumor after surgery in anamnesis	0	100	7.1	4.8
Polyneuropathy of lower extremities	68.5	36.4	17.9	11.9
Focal and meningeal symptoms	0	100	17.9	4.8
AWS	100	100	0	0
Increased GGTP indexes more than 10 times	78.5	70.0	0	0
Increased GGTP indexes 2-10 times	15.8	7.3	10.7	5.6
Increased AST indexes more than 10 times	65.3	68.3	0	3.4
Increased AST indexes 2-10 times	29.0	9.0	5.3	9.0
Increased ALT indexes more than 10 times	5.3	4.0	0	0
Increased ALT indexes 2-10 times	89.7	73.3	2.3	0

In the neurological status of the patients from the I group epiphenomena, hereditary tainted epilepsy, AWS, brain tumor after surgery in anamnesis were not revealed, however in 68.5% of cases, polyneuropathy of lower extremities has been detected. In 94.3% of the patients from the I group, high activity of blood serum ALT, AST, GGTP was registered.

In the neurological status of all the patients from the II-a group epiphenomena, hereditary tainted epilepsy, AWS, brain tumor after surgery in anamnesis were revealed. 77.3% of the patients had higher indexes of blood serum transferases, the GGTP level was maximally high, ALT-to a lesser degree, AST -2-4 times higher.

During collection of anamnestic data of the patients from the II-b group, it was detected that 73.2% of them had epiphenomena, 7.1% - CI in anamnesis, 3.6% - brain tumor after surgery in anamnesis, 19.4% - hereditary tainted epilepsy. Polyneuropathy of lower extremities was detected in 17.9% of the patients. 3-4 times higher activity of blood serum transferases (GGTP, ALT, AST) was registered in 10.7% of the patients.

In the III group, epileptic phenomena were detected in 23.8% of the patients, anamnestic record in epilepsy among 7.1% of the patients, craniocerebral injuries in anamnesis of 4.8% of the patients. During the clinical examination nontoxic polyneuropathy of lower extremities -were detected in 11.9% of them.

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